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Case Report

Fatal traumatic subarachnoid hemorrhage due to assault-related tear of the basilar artery

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ABSTRACT

Traumatic subarachnoid hemorrhage after blunt head injury or neck trauma most often occurs due to intracranial or extracranial vertebral artery rupture. A literature review confirms that subarachnoid hemorrhage related to basilar artery disruption is a rare event. Strong associations have been made between basal subarachnoid hemorrhage and relatively minor blunt force injuries to the face, head, or neck. Moreover, the degree of hemorrhage may appear striking and disproportionate to the external and internal evidence of injury. We present a case of an assault-related basilar artery tear causing fatal subarachnoid hemorrhage, despite minimal external injury. This report provides an overview of potential mechanisms accounting for vertebrobasilar system rupture, with application to our case.

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1. Introduction

Traumatic subarachnoid hemorrhage (SAH) has been a widely explored subject in the forensic pathology literature. Over the past century, increasingly strong associations have been made between basal SAH and relatively minor blunt force injuries to the face, head, or neck.[5] Several theories have been put forth endeavoring to explain the possible mechanisms behind traumatic vertebrobasilar system injury. The link between alcohol consumption and traumatic SAH has also been well documented in the literature. A majority of SAH cases associated with blunt head or neck trauma have cited the vertebral artery as the most common source of bleeding. The following case exemplifies a traumatic SAH fatality due to basilar artery disruption.

2. Case report

In the early evening hours, a 19 year old white male was assaulted in a mall parking lot. According to witnesses, the decedent received a punch to the face, following which he fell back against a wall and struck his head. He then walked away from the scene approximately 50 m, collapsed, and lost consciousness. A passerby noted that the decedent was apneic and pulseless, and promptly initiated cardiopulmonary resuscitation until the emergency medical personnel arrived. En route to the hospital, the decedent remained pulseless. Aggressive resuscitative efforts in the emergency room failed. There was no past medical history, and no previous history of either prescribed medication use or illicit drug abuse.

A medicolegal autopsy was performed 12 h postmortem. On external examination, a roughly crescentic abrasion was noted to the right upper forehead as the only external sign of trauma. There were extensive resuscitative findings including a single needle puncture site to the chest in continuity with the right ventricle of the heart. There was an associated hemopericardium of 80 ml of unclotted blood and bilateral small hemothoraces resulting from insertion of bilateral chest tubes. These played no material role in the decedent's death.

The internal examination revealed no natural disease process accounting for death. The only grossly apparent traumatic injury of the neck was soft tissue hemorrhage around the occipital muscle. No additional injuries to the neck were identified with a careful layered dry anterior and posterior neck dissection. The neck structures were excised, fixed, and imaged for radiologic examination. No fractures were identified. Additional radiography revealed no cervical vertebral body fractures or disc space abnormalities. Upon removal of the brain and associated vessels, basal subarachnoid hemorrhage was prominent. However, the extracranial vertebral and carotid arteries were fully intact. The brain and associated intracranial vessels was referred to a Neuropathologist experienced in Forensic Neuropathology for consultation and expert opinion.

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Microscopic examination revealed no evidence of natural disease in the vital organs including the heart, lung, liver, and kidneys. There was a small amount of acute hemorrhage noted within the left occipital muscle, as seen during gross examination. The accompanying Figs. 1 and 2 demonstrate hematoxylin and eosin staining and trichome staining of the rupture site. The sections demonstrated disruption through the full wall thickness of the vessel. There was no evidence of either microscopic vascular wall disruption, or atherosclerotic disease within the extracranial portions of the carotid and vertebral arteries.

The neuropathologic consultation reported bilateral symmetric SAH involving the frontal, orbital frontal, and anterior temporal regions. Additional SAH was noted over the brain stem and cerebellum, with no grossly apparent contusions to these structures. The Circle of Willis was of normal anatomic configuration with no aneurysms or gross evidence of atherosclerotic plaque formation. Microscopic examination of the cerebral cortex revealed patchy areas of acute hypoxic-ischemic injury, with no infarction. No other pathological process was evident in the cortex. On serial sectioning of the brain, there was extensive intraventricular hemorrhage into the fourth ventricle. Microscopic sections of basilar artery revealed focal areas of vascular wall disruption and fragmentation, with interspersed recent hemorrhage through the adventitia, media, and intimal layers. No cerebrovascular thrombus or vasculitic process was identified.

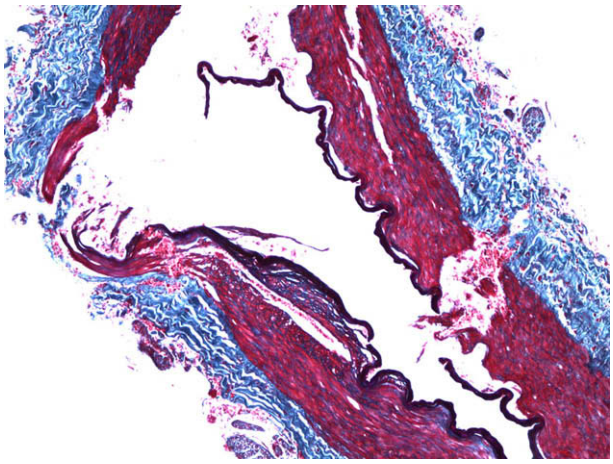

Fig. 1. Depicts the staining of the basilar artery using a trichrome stain to demonstrate the elastic lamina and shows transmural disruption of the wall of the vessel with attendance transmural hemorrhage.

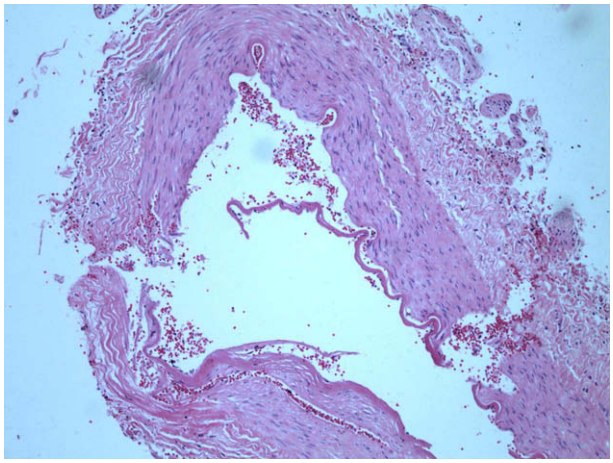

Fig. 2. A hematoxylin and eosin stained section of the basilar artery rupture site mag 100× showing transmural disruption of the vessel wall and attendant hemorrhage.

Postmortem toxicologic analysis positively identified cannabinoid metabolites and an alcohol level of 58 mg/100 mL (0.058% w/v) in the peripheral blood. This alcohol level is well below the limit for legally operating a motorized vehicle in Ontario, Canada (80 mg/100 ml). No other street drugs or drugs of therapeutic significance were identified.

Based on the combined postmortem and neuropathologic findings, the immediate cause of death was reported as a traumatic and extensive SAH secondary to a transmural and linear-type traumatic tear of the basilar artery.

An arrest was made and the assailant was charged under the Criminal Code of Canada with Manslaughter. During the trial, photographic evidence of a curvilinear injury to one of the assailant's fingers was presented and discussed. One interpretation offered by the prosecution was that contact with the decedent's dentition was the most likely cause of the assailant's injury. Upon completion of the trial, the assailant was convicted.

3. Discussion

It is well documented that traumatic SAH occurs with disruption of the vertebrobasilary arterial system. However, the exact origin of bleeding can be difficult to definitively demonstrate, particularly with tearing of small arterial branches.[5] In some cases, traumatic SAH may occur at a site of pre-existing aneurysm, while in most cases, there is no obvious site of vessel damage and the vasculature may be free of any natural disease.[4] If cerebrovascular injury is identified macroscopically or microscopically, a tear of the extracranial portion of the vertebral arteries is the most common finding.[1,2,4] If the source of the traumatic basal SAH is intracranial, the vertebral arteries are again most often implicated.[1] Interestingly, the intracranial vessels have thinner media and adventitial layers and could be more frequently fatally injured when compared to the thicker extracranial vessels.[1] The consistent finding in the literature is that traumatic basal SAH secondary to basilar artery wall disruption is rare.[1] One retrospective study examining 17 cases of fatal SAH due to trauma reported that only one of these deaths was attributed to basilar artery injury.[3] Their data indicate that 69% of cases expired within 1 h of injury, with a maximum survival time in one case of 45 h.[3]

There is strong consensus in the literature that traumatic basal SAH can occur after negligible blunt force injury to the face, head, or neck.[1–6,11] The degree of hemorrhage may appear striking and disproportionate to the external or internal evidence of injury.[5] Therefore, a precise mechanism of injury likely plays a more significant role in traumatic SAH rather than the magnitude of the traumatic force.[3] There are several well-cited theories in the literature attempting to explain potential mechanisms of intracranial arterial injury. One explanation is that vertebrobasilar tears occur due to a rotational acceleration of the head, resulting in a transient period of brain oscillation.[1,2,4,5] A second theory proposes that such tears can occur secondary to a brief but severe increase in intra-arterial pressure resulting from a blow to the carotid artery.[1,2,4,5] Another popular theory suggests that acute hyperextension of the neck endangers the basilar and vertebral arteries due to anatomic location.[1,2,4,5] Sudden neck hyperextension can occur with midline blows to the face or jaw, such as during assault, or during a collision in which the chin strikes the steering wheel.[1,2] Blunt force injury to the posterolateral or lateral neck could be an additional mechanism, resulting in disruption of the vessels at the base of the skull.[2,7] According to some authors, the risk of neck hyperextension causing death is increased in individuals over 40 years of

age.[6] The most likely explanation for this is an age-related loss of cervical muscle tone or degeneration of the cervical spine.[6] Nevertheless, traumatic SAH is far more common among young, healthy, and intoxicated men, and is rarely seen among the elderly population.[2]

Several authors have cited an additional mechanism explaining increased risk of fatal SAH. They postulate that intense emotional excitement associated with a traumatic event such as an altercation, could result in arterial hypertension, thereby placing the vertebrobasilar system at higher risk of rupture.[2,4] This may be plausible if significant underlying congenital vessel wall weakness is present. However, experiments have determined that pressures up to 1520 mmHg would be required to cause cerebral artery rupture, thus making this mechanism highly unlikely.[2] Dowling and Curry[4] suggest that the trauma itself, rather than excitement-induced hypertension, would be a more important causative factor.

Another factor often discussed in the literature and believed to be associated with traumatic SAH, is alcohol consumption. Several sources have noted that 75–87% of traumatic basal SAH cases reviewed were either under the influence of alcohol or intoxicated.[1,3] Harland et al.[3] determined a mean blood alcohol level of 213 per 100 mL in the cases they reviewed. One study by Blaha et al.[8] demonstrated that a state of mild alcohol intoxication resulted in increased cerebral blood flow by up to 24%, due to higher middle cerebral artery flow velocities. They also note that minor or severe head injury can impair normal cerebral vascular tone and autoregulation.[8] Alcohol is known to promote local vasodilation in the cerebral circulation, and has been shown to exert an anti-vasospasm effect in animal studies.[9] It is possible that mild or significant alcohol consumption may render the cerebral arteries more susceptible to trauma-related damage and rupture, and may cause a more rapid death due to local vasospasm inhibition.[3] Alcohol is also known to decrease coordination and reaction time, which could result in more severe shearing force should an unanticipated blow to the head occur.[1] Interestingly, marijuana use has also been associated with increased cerebrovascular flow velocity compared to controls.[10]

In the present case, the decedent suffered a linear and transmural basilar artery tear due to shearing forces occurring with sudden neck hyperextension. He succumbed to this injury within minutes of the assault. There was no evidence of significant blunt force trauma on external examination, and the source of the SAH was only apparent on microscopic examination. Traumatic SAH may occur with minor blunt trauma to the face or neck, despite having a vertebrobasilar arterial system free of disease.[5] It is therefore important that the Forensic Pathologist painstakingly examine the vertebrobasilary system to either rule in or rule out whether the etiology is related to a traumatic lesion or aneurysm. Meticulous postmortem removal of the brain, Circle of Willis, and vertebral arteries is vital. Technical care must be used to minimize the likelihood of artifactually inducing injury since the forces during brain removal may result in inadvertent arterial tears.[5] The source of bleeding can be obscured by cutting the vertebral arteries too close to the basilar artery origin, thereby missing potentially important clues to the mechanism of injury.[4] Djokic et al.[5] recommend opening the basilar artery longitudinally and sectioning it in the same way to improve visualization of posterior wall changes. Postmortem angiography can also aid in establishing the site of hemorrhage prior to removal of the brain and vessels, however injection of contrast may also induce artifactual tears.[4] Many centers do not routinely perform vertebrobasilar system angiography for this reason. Radiographic examination of the cervical spine to confirm or exclude fracture of the transverse processes is also recommended in standard autopsy protocols in cases of traumatic SAH.[4]

In addition to confirmed microscopic evidence of a rare isolated basilar artery tear injury in the present case, other postmortem findings were consistent with a neck hyperextension injury. The extravasation of blood into the occipital muscle supports a neck hyperextension injury.[5,6] Severe hyperextension may also result in intraventricular hemorrhage extending into the fourth ventricle.[6] This finding was present in this case. Although hyperextension can produce brain stem injury such as pontomedullary avulsion, this was not found in this case.[6] Traumatic SAH should be suspected if there are obvious injuries to the ear, upper neck, or parotid region.[3] The decedent did not sustain such injuries. The assailant in this case demonstrated injury giving a clue to the point of contact with the decedent. Finally, the decedent was noted to have low serum alcohol and marijuana levels at the time of assault. Based on the above discussion, these substances may have made him more susceptible to the tear, and may have contributed to the rapidity of his death.

Conflict of Interest

None.

Funding

None.

Ethical Approval

This report satisfied the ethical requirements of the Research Ethics Board of Hamilton Health Sciences/Faculty of Health Sciences, McMaster University.

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